liver and renal functions were normal. Arterial ultrasound and computed tomography scan of the legs confirmed the presence of a voluminous popliteal aneurysm (diameter: 50 mm, length: 100 mm), with partial mural thrombus and a residual lumen of 15 mm. Indium 111-labeled platelet scintigraphy showed increased accumulation of radioactivity over the aneurysm (Fig. 1). No surgery was performed because of the patient's advanced age.

Increased fibrin split products may be present in as much as 40% of patients with aortic aneurysm. A more severe consumption coagulopathy with thrombocytopenia, however, is observed in only 4% of cases [1]. Usually, these aneurysms are extensive, involving the thoraco-abdominal aorta [1–3], and the pathogenesis of the consumption coagulopathy appears to be a continuous dynamic process of intravascular coagulation with platelet consumption and secondary fibrinolysis [1,4]. In this patient, no aneurysm was detected on the thoracoabdominal aorta, and the indium-labeled platelet scintigraphy showed that the coagulopathy initiated in the popliteal aneurysm. To our knowledge, this is the first time such an observation has been made, but this popliteal aneurysm was unusually large and thrombosed. This observation suggests that size and shear flow conditions inside the lumen, rather than location of the aneurysm, might be critical for the development of chronic consumption coagulopathy.

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Persistent Polyclonal B Lymphocytosis

To the Editor: We report a case of persistent polyclonal B lymphocytosis which presents, in contrast with others communicated in the literature, certain differences.

A 35-year-old woman was admitted to hospital 2 years after presenting with a persistent lymphocytosis whose cause had not been investigated during this period. Prior medical history disclosed only psoriasis and duodenal ulcer. She was a heavy smoker (20 cigarettes/day) and had complained of asthenia during the preceding 4 months. Physical examination was negative. Peripheral blood showed: hemoglobin 130 g/l; white blood cell count 11×10^9/l (24% neutrophils, 70% lymphocytes; 3% monocytes; 3% eosinophils); platelets 203×10^9/l. Morphologic study of the lymphocytes showed 60% atypical and 5% binucleated forms (Fig. 1). No evidence for an active or persistent viral or rheumatologic disease was found (only evidence of a previous Epstein-Barr virus [EBV] infection was detected, viral capsid antibody IgG 1:640). Immunophenotyping showed that the lymphocytosis was of the polyclonal B-cell type: CD19+, CD20+, CD21+, CD22+, and both kappa and lambda light-chains were expressed, with a ratio of 6/11. Serum immunoglobulins revealed decreased levels of IgA and IgG and high levels of polyclonal IgM (7.37 g/l). Bone marrow aspirate showed slight lymphocytosis (30%), with 10% atypical forms. The patient was followed-up for 4 years without any change in the physical examination,

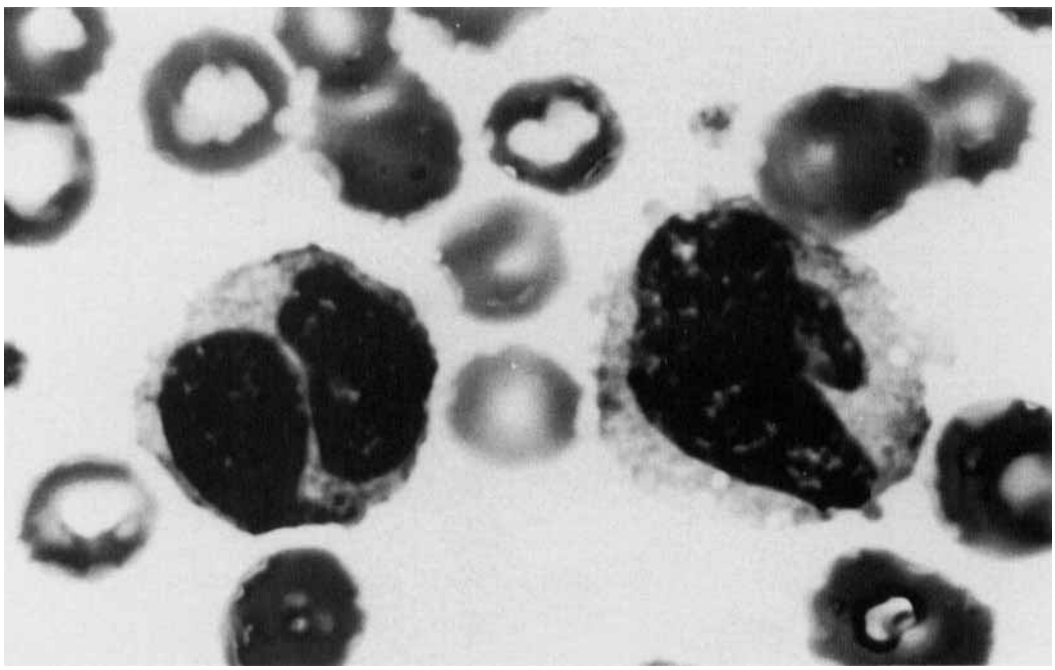

Fig. 1. Binucleated lymphocyte in peripheral blood.

features observed in peripheral blood, or serum immunoglobulin levels (IgM peak 12.7 g/l). After this period, she ceased smoking and a reduction in the percentage of lymphocytes in peripheral blood was observed (mean 48%), though the abnormal forms persisted (1%). In the same way a reduction in the level of serum IgM was observed (mean 4.85 g/l). HLA-typing performed later showed that our patient was negative for HLA-DR7. A study of her relatives (parents and sister) failed to disclose similar abnormalities (binucleated lymphocytes or increased IgM).

Since this syndrome was first described by Gordon et al. [1], several possible etiologic mechanisms have been suggested, including: (a) genetic predisposition [2–4], based on HLA-DR7 positivity in most patients and the presence of binucleated lymphocytes in their relatives, though the complete syndrome may be absent; (b) cigarette smoking [4,5]: a great number of these females were smokers, and in a patient reported by Carstairs et al. [5] a cure of the lymphocytosis was observed when she stopped smoking; and (c) viral infection: Chow et al. [6] demonstrated EBV DNA in lymphocytes of two patients suffering from this syndrome. In our case, the patient was HLA-DR7-negative and none of her relatives presented with similar abnormalities in peripheral blood, making the genetic hypothesis improbable. Marked reductions in serum IgM levels, in the absolute number of lymphocytes, and in the percentage of binucleated forms were observed when the patient stopped smoking. Finally, the hypothesis of EBV as the cause of this syndrome cannot be excluded since a prior infection has been demonstrated in the patient. In our opinion, this polyclonal B-cell lymphocytosis is a disorder which should be grouped with several others with differing clinical expressions. Some patients have presented with splenomegaly or adenopathies [1–4], which have still to be clearly defined.

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Thrombin–Antithrombin III and Prothrombin Fragment 1.2 Levels in Early Respiratory Distress Syndrome

To the Editor: Disseminated intravascular coagulation (DIC) is frequently encountered in preterm neonates with advanced respiratory distress syndrome (RDS) [1]. However, we previously reported normal plasma fibrinogen, antithrombin III (AT-III), protein C, and tissue plasminogen activator but lower D-dimer and higher plasminogen activator inhibitor (PAI) levels within the first few hours of life in preterm infants who later developed RDS compared to control group [2]. These changes in D-dimer and PAI levels are probably related to abnormalities in the fibrinolytic mechanism due to lung damage and local platelet activation in RDS. Therefore, we studied some of the more specific DIC parameters in these patients to evaluate the coagulation disorders in detail.

TABLE I. TAT and F1.2 Levels in Preterm Infants With or Without RDS (Mean ± SD)

	Controls (n = 20)	Infants with RDS (n = 15)
Gestational age (weeks)	32.1 ± 2.1	32.4 ± 1.7
Birth weight (g)	1614 ± 439	1851 ± 602
TAT (μg/l)	78.12 ± 72.75	64.83 ± 69.91
F1.2 (nmol/l)	9.02 ± 7.37	9.87 ± 5.67

Abbreviations: TAT, thrombin/antithrombin III complex, F1.2, prothrombin fragment 1.2; RDS, respiratory distress syndrome.

Theoretically, the specific detection of thrombin should be suitable for use in the active state of DIC. However, thrombin is very rapidly bound and thereby inactivated by its main physiological inhibitor, AT-III. For this reason, a more direct method to evaluate the thrombin level is to measure the thrombin/antithrombin III complex (TAT) formed with AT-III. Patients with DIC are found to have elevated concentrations of TAT [3–6]. In practice, thrombin can, therefore, be measured only indirectly, by assaying the cleavage products of prothrombin released during the conversion to thrombin (prothrombin fragment 1.2 [F1.2]) or by assaying activation products of substrates of thrombin (fibrinogen, fibrin degradation products, fibrinopeptidase A, etc.). The assays commonly used—i.e., prothrombin time, activated partial thromboplastin time, fibrinogen or fibrin degredation products—are insensitive and nonspecific in the diagnosis of DIC [7]. However, F1.2 is a polypeptide released from prothrombin during its activation to thrombin. F1.2 is a biological marker of the thrombin generation, and it has been demonstrated to correlate with the thrombotic risk associated with certain patient populations [8–10].

Therefore, we studied serum TAT and F1.2 status in 35 preterm infants with or without RDS in the first few hours of life. All neonates received vitamin K1, 1 mg, intramuscularly upon delivery. Blood samples for TAT (enzyme immunoassay method; Enzygost TAT micro, Behring, Germany) [6] and F1.2 (sandwich-type ELISA method; Enzygnost F 1 + 2 micro, Behring) [10] testing were obtained from a peripheral vein within 6 hr after birth and mixed with 3.8% trisodium citrate according to their hematocrit levels. The tubes were centrifuged at about 3,000 rpm for 10 min within 30 min of collection. The plasma was stored at −20°C less than 1 month before the procedure.

Among 35 infants, 20 who were in stable clinical condition served as the control group. Fifteen developed RDS, which was considered to be present if all of the following diagnostic criteria were fullfilled: symptoms of respiratory distress within 1 hr after birth and present for at least 24 hr, respiratory support including mechanical ventilation, and typical findings on lung x-ray and arterial blood gas analysis. None of the infants with RDS had any other disease. The mean plasma TAT and F1.2 levels were found to be similar in the two groups ($P > 0.05$ by Mann-Whitney U test; Table I).

These relatively more specific parameters (i.e., TAT and F1.2) of DIC support our previous hypothesis, as DIC is not a prominent event in early (or developing) RDS [2]. For this reason, further studies are needed to show the other abnormalities in the hemostatic system and their pathogenic significance in RDS. The main limitation in these studies is the ethical dilemma of attaining enough blood samples in the first few hours of life to evaluate the various parameters of coagulation and the fibrinolytic system simultaneously. The accumulated findings from different centers in the literature will be useful in reaching a reliable conclusion.